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THE RENAL ASPECTS OF HYPERTENSIVE-VASCULAR DISEASE*

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Physicians who graduated before the first World War will remember the confusion surrounding the subject of hypertension and its relationship to kidney diseases and arteriosclerosis. Richard Bright had sought a reason for the hypertrophy of the heart which he found in so many of his patients with kidney disease. He suspected an altered quality of the blood or an increased peripheral resistance. The celebrated paper by Gull and Sutton in 1872 on "Arterio-capillary Fibrosis" called attention to "the morbid change in the arterioles and capillaries as the primary and essential condition of the morbid state called chronic Bright's disease with contracted kidneys." Clinical sphygmomanometry (von Basch) enabled Allbutt in England, Huchard in France and a little later Janeway in America to make some noteworthy advances in the understanding of "nonrenal" hypertension. Prior to their observations, which occurred about the turn of the present century, it was thought that arteriosclerosis was responsible for hypertension and that all such patients would die of chronic kidney disease. Allbutt, however, called attention to a large group of individuals in whom hypertension existed without any evidence of kidney disease, and this process, widespread in the civilized world, has come to be known as essential hypertension.

There is little need to emphasize the importance of hypertension. According to the statistics of the Metropolitan Life Insurance Company every other individual in the United States past the age of 50 years dies of cardiovascular-renal disease. From other sources we have evidence that probably half of these deaths are due to essential hypertension, that is that almost one-quarter of all people past the age of 50 years die of the effects of hypertension on one or another of the vital organs. Thus essential hypertension becomes the gravest problem of middle adult life, not even excepting cancer. The hypertension arises from arteriolar vasospasm (whatever its cause may be) and after it has continued for years a compensatory reaction in the arterial system results in a diffuse hyperplastic vascular sclerosis, which, in turn is eventually responsible for failure of such organs as heart, brain and kidney.

This is a very different process from the senile sclerotic changes which occur in large vessels such as the aorta and its main branches. It is unfortunate that the term arteriosclerosis is used for both processes. Even the introduction of the modified term arteriosclerosis to fit the hypertensive-vascular process mentioned above is not satisfactory. The similarity in the two terms leads to confusion. It has been generally accepted that essential hypertension is the best designation for the first process and hypertensive-vascular disease for the vascular reaction that results. The old term atherosclerosis is again becoming popular for the second group of disorders and thus we may permit arteriosclerosis to be an inclusive term comprising both forms.

Now our first difficulty enters. While it is true, as stated above, that these are independent processes and can exist alone, that is, hypertensive-vascular disease without atherosclerosis and atherosclerosis without hypertension, nevertheless, they may coexist and frequently do. This is quite natural because hypertensive-vascular disease is one of the commonest disorders of middle life and atherosclerosis is the commonest disorder of late life. Consequently individuals with hy-

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pertensive-vascular disease live into the age period when atherosclerosis develops. Furthermore, there is some evidence that the progression of atherosclerosis may be accelerated by the hypertensive process.

What is the relation of these vascular disorders to kidney disease? Hypertensive-vascular disease, through its narrowing effects on the afferent arterioles of the kidney, may gradually lead to greater or lesser degrees of glomerular obliteration with tubular atrophy and interstitial fibrosis. This is the condition now often referred to as nephrosclerosis, the so-called primary contracted kidney of older writers and one of the varieties of chronic interstitial nephritis. In regard to the latter term it is interesting to recall the great controversy between pathologists and clinicians which gave rise to the false dictum "there can be no correlation between clinical and pathological findings in kidney disease." Processes such as atherosclerosis, chronic glomerulo-nephritis and pyelonephritis produce a scarred kidney which more or less closely resembles the organ resulting from hypertensive-vascular disease. Therefore, the older pathologists called them all "chronic interstitial nephritis" but were forced to admit that the gross scarring of the kidney at the post mortem table gave no indication of the number and condition of the functionating units that were present during life. Hence, they could not correlate their findings with the clinical picture.

We know that hypertensive-vascular disease may encroach sufficiently on kidney substance (nephrosclerosis) to lead to kidney insufficiency and uremia but that this occurs in only about 10 per cent or less of individuals with essential hypertension. The reason is that before the kidneys are affected to a degree incompatible with life a more vital organ (heart or brain) has succumbed. As Allbutt expressed it, "the overstrained vessels in the brain will burst, the laboring heart will suffer defeat, before the symptoms of uremia are likely to make their appearance." Malignant hypertension, to be discussed, ends in renal failure more frequently.

Atherosclerosis affects the kidney to a minor degree so far as clinical results are concerned. That is, the process in the larger vessels may lead to gross scarring of the kidney but so few renal units are involved that the effects usually are of little consequence. The slight albuminuria that may occur is of no clinical significance; too often, in the past, it has been accepted as evidence of progressive kidney disease that would lead to uremia. Again Allbutt succinctly stated the case for the senile arteriosclerotic kidney when he said "it is a scarred but not a corrupt kidney, sufficient for the smaller life of an elderly man."

Nephrosclerosis, then, is the specific end-result of the renal reaction to the hypertensive-vascular process. It leads to death from renal failure in 10 per cent or less of all instances of essential hypertension. Nevertheless, so common is essential hypertension that this 10 per cent is still much more important than glomerulonephritis as a cause of death, and vital statistics that give such prominence to "nephritis" as a cause of death, are derived not only from the deaths caused by glomerulonephritis but also by nephrosclerosis and other conditions which are frequently included in the term cardiovascular-renal disease. It has been stated and should be emphasized that these diseases while treated as a group for statistical purposes, must be dealt with separately if we are to advance in our knowledge concerning them.

It has been suggested that there are two main avenues of approach to chronic kidney disease; one is vascular and degenerative, embracing the term nephrosclerosis, and the other is inflammatory and embraces the terms glomerulonephritis and pyelonephritis. Renal failure may be brought about by either method. But whatever the cause of the renal impairment, the clinical manifestations may be the same; as the kidney fails its concentrating ability diminishes and waste substances accumulate in the blood. Eventually the clinical state known as uremia occurs.

Not all patients with cardiovascular-renal disease die in ways indicated above because occasionally it seems as if there is an almost simultaneous disintegration of vital organs and, of course, it is possible for intercurrent infection to carry away a subject who has been weakened by the gradual encroachment

on his vital organs by these insidious processes.

ESSENTIAL HYPERTENSION

The most important aspect of this whole problem is the etiology of so-called essential hypertension which stands in the background of the widespread vascular disease and eventual organ failure.

As suggested in the previous discussion we were just beginning to feel that we had at last straightened out the relationships of essential hypertension, kidney disease and arteriosclerosis when the work of Goldblatt upset our recently achieved complacency. We had labored for more than a quarter of a century to free ourselves from the idea that essential hypertension was of renal origin, and now Goldblatt is leading us right back to the renal origin of essential hypertension in a brilliantly conceived and beautifully executed series of experiments. He has demonstrated for the first time the experimental production of a persistent hypertension in animals which has all the characteristics of essential hypertension in man, and he has produced this condition simply by the application of an ingenious silver clamp to the renal artery by means of which varying degrees of renal ischemia are produced. Within a short time persistent hypertension follows which varies in direct proportion to the degree of renal ischemia. Moreover, vascular and organ changes similar to those which result from essential hypertension in man are produced. As in man these vascular changes are especially well observed in the retina.

As a result of this work, many questions regarding the etiology and pathogenesis of hypertension arise—is the hypertension produced through the intermediation of the nervous control of the kidneys, that is, nervous reflexes from the ischemic kidney, causing a generalized vasoconstriction, or is it a humoral mechanism—is the ischemic kidney producing some effective substance which acts through the blood stream to produce a generalized vasoconstriction?

The first question seems pretty well answered in the negative. No matter what is done to the sympathetic nervous system of the dog, once the renal clamps are applied nothing will lower the blood pressure. In fact, the whole sympathetic nervous system can be removed and the hypertension persists. Once the importance of the sympathetic nervous system in the development of experimental hypertension had been excluded it was necessary to determine the possible relationship of certain glands of internal secretion. While I will not take time to cite these experiments in detail the work of Goldblatt and others suggests that the endocrine glands are not essential for the development of hypertension associated with renal ischemia but that they are important in determining the degree of response.

Now the question which interests us—if we are to compare this process to the problems of human hypertension—is this: is essential hypertension in man also due to renal ischemia and if so what is the mechanism? You will remember that we had previously convinced ourselves that the unimpaired renal function of essential hypertension meant that the kidneys were blameless. Now we have learned to differentiate this excretory function of the kidney from the circulatory function and to realize that the latter may be impaired while the former is normal. In this connection the very recent experimental work of Page and his associates must be mentioned. They feel that the stimulus to renal hypertension is not renal ischemia as such, but rather a reduction in pulse pressure within the kidney. A very ingenious model demonstrated this mechanism at the scientific exhibit of the last meeting of the American Medical Association. The renal ischemia which arises in the isolated kidney, is the result and not the cause of the process which leads to the development of hypertension, according to these investigators. Regardless of this point, however, evidence is accumulating that suggests the renal origin of essential hypertension in man in a manner comparable to that of experimental hypertension in animals. Pathological studies find the arterioles of the kidney involved to a much greater extent than are arterioles throughout the rest of the body in those patients with essential hypertension who have been studied at autopsy. Moreover, it is a striking fact that although arteriolar disease occurs in the organs of the

individuals without hypertension it is almost never found in their kidneys while in hypertensive individuals, in addition to the vascular disease of other organs, the kidneys almost never escape.

The problem today is essentially comparable to the older concept which disturbed the medical world for so many years concerning the relationship of hypertension and arteriosclerosis except that it has been narrowed down to the kidney. As previously stated it was originally held that arteriosclerosis caused hypertension; subsequently the pendulum swung in the opposite direction and it was concluded that hypertension caused arteriosclerosis (diffuse hyperplastic sclerosis). For a number of years we enjoyed considerable security in this conclusion until the work of Goldblatt led us back to the renal origin of hypertension. And now the studies of Moritz and Oldt raise the old question again—does hypertension result because of a primary renal arteriosclerosis or is it the other way around—are the renal arterioles peculiarly vulnerable to the effects of hypertension and hence show such a high incidence of arteriosclerosis in hypertensive subjects? Moritz and Oldt hold the former view and feel that their study lends pathological confirmation to Goldblatt's work so far as human hypertension is concerned.

I am prepared to believe that interference with the circulatory function of the kidney is the pathogenetic basis for hypertension in man but I am not at all persuaded that structural (arteriosclerosis) changes are primarily responsible. It seems to me much more likely that they are secondary to functional vasospastic changes such as we can so clearly observe in the retina. In this regard it is noteworthy that there is a very close correlation between retinal and renal arteriolosclerosis which emphasizes the value of ophthalmoscopy in the study of hypertensive-vascular disease.

Significant in regard to the mechanism just discussed is the work of Homer Smith, Goldring and their associates who employed diodrast, phenol red, and inulin clearances to determine renal blood flow in normal and hypertensive human subjects. They appear to demonstrate that there is a diminished renal blood flow in hypertensive subjects despite normal renal function. Furthermore, as Page also suggests, the renal blood flow appears to be controlled predominantly by the efferent glomerular arterioles.

Finally, some evidence for the renal origin of hypertension in man comes from a few cases that have now been reported in which unilateral renal disease seems to have been responsible for hypertension—at least the hypertension has promptly disappeared when the diseased kidney has been removed. May I say in passing that I think we will be disappointed in our efforts to cure hypertension by the removal of unilaterally diseased kidneys. I cannot take time to discuss this in detail but probably the undetermined vascular disease of the supposedly healthy kidney will perpetuate the hypertension. The new emphasis on the renal origin of hypertension has been so exciting that we are in danger of being led astray from a practical treatment standpoint. This is not saying that the new orientation has not been good for us—urological investigation has assumed a new importance in the study of patients with hypertension—and vigorous efforts are being made to break down so-called essential hypertension into various groups but these efforts are more determined than successful. Unfortunately, we are still a long way from understanding the cause of the majority of cases of hypertension.

PRESSOR SUBSTANCES

A pressor substance had long been suspected as the cause of essential hypertension. Coincidentally with the observations of Goldblatt and his associates just referred to, there has been a renewed interest in this question. Recent studies indicate that a thermolabile, protein-like substance which can be isolated from the renal cortex is present in the normal kidney, but is found in increased amounts in the ischemic kidney of animals. It has been shown to exert a marked pressor effect upon the blood pressure. Thus, there is evidence in favor of a chemical stimulus of renal origin, exerting a generalized pressor effect upon the blood vessels, and acting independently of the central nervous system.

PSYCHE AND HYPERTENSION

It is generally admitted that psychic fac-

tors play an important part in essential hypertension. Thus it is always emphasized that we must allow for the emotional element in individual blood pressure readings. It is also well known that rest and reassurance play a large part in the medical management of hypertensive patients both in relief of symptoms and in reduction of the blood pressure level. The early symptoms of hypertension are often exactly those of a psychoneurosis and the relationship of emotional stress to the onset of hypertension and to the anxiety which is frequently responsible for the aggravation of existing hypertension are well known. Personality study often reveals deep-seated conflict which stands in close relationship to this anxiety. Therefore it may be said that in spite of the organic nature of experimental hypertension the central nervous system is not absolved as a factor in the etiology of hypertension in man. It is conceivable that a disturbance in the circulatory function of the kidneys could have its origin in impulses of central nervous origin.

I have attempted to discuss only some of the more recent studies which relate the kidney to the pathogenesis of hypertension. Etiological factors such as heredity, race and geography I have considered in some detail elsewhere[1] and shall not take the time to repeat now.

CLINICAL PICTURE

It is well known that hypertension may exist for a long period without any symptoms whatever. When symptoms finally appear, they frequently arise from the organ or system that is bearing the brunt of the hypertensive vascular disease process. Thus the symptoms may be related entirely to the heart or neurological symptoms may predominate if the brain is chiefly affected. In terminal stages evidences of renal failure may dominate the clinical picture. However, as previously stated, many of the early symptoms are identical with those that are seen in psychoneurosis. Headache, dizziness, tinnitus, insomnia and fatigue are prominent symptoms and often are out of proportion to the amount of physical disease that is present.

The problem often met in practice is as follows: an individual of middle age applies for life insurance; high blood pressure and perhaps albuminuria are discovered. Insurance is refused and, frightened or angry, he seeks the advice of his physician. General physical examination with special attention to the cardiovascular system, particularly in respect to the size of the heart, the condition of the peripheral vessels, including those of the retina, plus tests of kidney function*, will enable one to establish a diagnosis in the majority of instances. The problem resolves itself, with certain very unusual exceptions, into the differentiation of glomerulonephritis from hypertensive-vascular disease. Usualy, it will be found that the patient is suffering from essential hypertension and the end result, perhaps 5 to 10 years away, will be cardiac failure in about 50 per cent, intracranial accident (hemorrhage or thrombosis) in about 25-35 per cent and renal failure in probably less than 10 per cent. Other accidents and incidental infections account for the remainder of deaths.

If the end result is to be renal failure, the patient, usually over a period of years, will show gradual loss of concentrating power as determined by specific gravity tests of kidney function. The final event, renal failure ending in uremia, will be indicated by the onset of retinitis and progressive nitrogen retention. This change may come about with considerable rapidity. That is, patients who present the usual clinical picture of benign hypertension may quite suddenly complain of severe headaches, show a higher level of blood pressure, especially the diastolic element, increased eye signs (the result of actual retinitis), loss of renal concentrating power, non-protein nitrogen retention and rapid progression to death. This acceleration of the hypertensive process has been called by Fishberg the malignant phase of essential hypertension.

MALIGNANT HYPERTENSION

Malignant hypertension and malignant nephrosclerosis are terms that have been used synonymously, but nephrosclerosis ought to be reserved clinically for those instances in which definite evidence of impairment of renal function exists. When essential hyper-

*Concentration tests, urea clearance, and intravenous urography are commonly performed. Unfortunately there are no simple methods available for testing the circulatory function of the kidneys.

tension occurs in young persons it is apt to run a more severe course. Volhard has said, "the younger the patient and the higher the diastolic pressure, the greater the danger of a malignant course." Such patients are much more likely to die of renal failure than patients with the clinical picture of ordinary benign hypertension. So definite is the clinical picture in this group of cases that the condition is looked upon by many as a distinct disease, quite different from benign hypertension. Keith, Wagener and Kernohan, who described a characteristic retinal picture for this condition, are responsible for the name malignant hypertension. The patient is usually young (less than forty); the pressure is very high, 250/160 is not unusual; and the retinal picture is characterized by arteriosclerosis of the hypertensive type, vasospastic retinitis and edema of the discs. The condition runs a rapidly fatal course (two years or less) and often ends in uremia, although there may seem to be an almost simultaneous disintegration of vital organs. The differentiation of malignant hypertension from glomerulonephritis often presents great difficulties. Also, it may not be amiss to mention that the swelling of the optic disc may resemble choking of the disc, and this finding in the presence of intense headache, nausea and vomiting may be so suggestive of brain tumor that a number of such patients have actually been operated upon. Pathologically, the kidney presents a characteristic picture with necrosis of afferent arterioles, "necrotizing arteriolitis," and consequent widespread obliteration of glomeruli, which probably accounts for the rapid onset of renal failure.

I agree with many others that it is best to consider this process simply as a severe form of essential hypertension which may arise quite suddenly, usually in younger people, or that may engraft itself at any time on the benign form. The fundamental mechanism seems to be the same as in benign hypertension, namely, a vasospastic process of probable renal origin. The difference in the two processes appears to be simply a question of the degree of intensity. The process is accelerated in the malignant form. It is something like the problem of acute and chronic leukemia, which also are very different in their clinical aspects; so different in fact that many have maintained that they are distinct diseases. Carrying the analogy further, acute leukemia may even engraft itself on and terminate chronic leukemia.

Why the kidneys suffer a greater injury in the malignant form of essential hypertension is a difficult question to answer. For some reason, renal arterioles seem more vulnerable with respect to this severe vasospastic disorder and hence the kidneys are the first organs to be destroyed.

TREATMENT

The treatment of nephrosclerosis, that is, the renal part of the picture of hypertensive-vascular disease, does not differ from the symptomatic management of the progressive loss of kidney function in glomerulonephritis. There is no specific drug or organ extract that will stop the process. The principles of management are the same; to maintain nutrition and supply an adequate amount of fluid for compensatory polyuria. When it comes to the question of renal failure occurring with malignant hypertension, the treatment again is the same as in uremia arising from glomerulonephritis. More important than the consideration of these end stages, however, is the problem of the management of the essential hypertension which precedes nephrosclerosis. While it is not the purpose of this paper to deal with treatment in any detail, I must refer to a few principles of management in an effort to give a comprehensive picture.

Let us take again the example of a middle-aged man refused by a life insurance company because of high blood pressure. His physician rules out glomerulonephritis and decides that the patient is suffering from essential hypertension. Then, all too frequently, attention is concentrated on the effort to "bring the blood pressure down." The patient demands to know the blood pressure figure; on each visit to the physician he waits with anxious concern to hear the latest reading and frequently he has ideas of "stroke," "heart failure" or "Bright's disease" in the back of his mind.

Just what has been done to this poor patient in the effort to "bring his blood pressure down?" Because of an ill-founded idea that protein is responsible for hypertension and

kidney disease he is denied meat and eggs, especially red meat, which for some reason is looked upon with particular dread. Then his diet is rendered even more unpalatable by the withdrawal of salt. One would sympathize with this half-starved victim of good intentions except that he probably would not be able to eat anyway, his teeth having been removed on the theory that focal infection has something to do with hypertension. Even before this period he has sacrificed his tonsils and has had his sinuses punctured because of the same theory. In case he actually had been able to eat some solid food, in spite of these previous therapeutic measures, the slight colonic residue was promptly washed out by numerous "colonic irrigations," especially during the period when the theory of autointoxication was enjoying a wave of popularity. To add to his unhappiness he may be told to stop work and exercise, and of course, is denied alcohol and tobacco as well as coffee and tea. And now to cap the climax of his difficulties, the unfortunate person with hypertension seems about to fall into the clutches of the neurosurgeon, who is prepared to separate him from his sympathetic nervous system.

PREVENTION OF HYPERTENSION

While it is true that we do not know the cause of essential hypertension, we do know enough about the nature of this process to plan a more rational management. Evidence is increasing concerning the deep-seated constitutional nature of this disorder, and superficial efforts to lower blood pressure and one-sided approaches to the problems must be condemned.

What are the stressful or vasospastic agents, the effects of which we must try to minimize? First of all, there is the question of obesity, because the build and weight of the individual have been shown to be of real importance in the etiology of hypertension. I cannot discuss all of the dietary fads that have prevailed in regard to the treatment of essential hypertension. The question can be dismissed by saying that in spite of the dread of protein, and red meat in particular, there is no evidence to prove that protein bears any relation to the cause of essential hypertension and the same is true of salt, that is, sodium chloride. It is the total diet which is important and then only in relation to obesity.

A second and even more important consideration is the question of the toxemia of pregnancy. Recently it has been shown that almost one-half of all women who have toxemia of pregnancy will develop hypertensive-vascular disease or chronic nephritis within a period of five years. This seems to be true of the milder degrees of toxemia as well as the more severe forms. The clinical deduction is obvious—if we are to prevent hypertensive-vascular disease and kidney disease we must make less effort to nurse these women through their toxemias and must make more effort to interrupt their pregnancies or induce labor. Ophthalmoscopic study is going to prove of inestimable value here, and the obstetrician will have to learn its value and use just as he has that of the blood pressure determination.

FOCAL INFECTION

When the chapter on focal infection is finally written, we will find that we have been overcredulous in regard to this concept in the etiology of many chronic diseases. I refer particularly to the question of foci of infection as a cause of essential hypertension. There is no evidence that there is any real relationship between them, and this is just as true in regard to treatment. The removal of so-called foci of infection will not cure essential hypertension. This does not mean that foci of infection should not be removed from hypertensive or prehypertensive people, but only for the same purpose that they are removed in other people.

Tobacco seems definitely to be a vasospastic agent in some persons and had better be denied to hypertensives and candidates for hypertension.

The last consideration for discussion is the question of psychic factors in relationship to essential hypertension. When all is said and done, essential hypertension seems to be the result of the reaction of a predisposed individual to his environment. Therefore, it seems reasonable to study this individual in relationship to his environment rather than to throw up our hands hopelessly because of his predisposition. This calls for physical and mental hygiene in its best sense. Mental hygiene does not mean, as is so often the case,

that the physician asks the patient: "Are you worried about anything?"—and is satisfied with a negative reply. This, by the way, is the same physician who will say to the hypertensive patient: "Stop worrying," or "Go home and take it easy." The one approach is just about as ineffective as the other. There is only one method that has any merit and that is to encourage the patient to talk about himself as a person rather than as a medical case. This must be done by skillfully guiding conversation rather than by asking direct questions. This will often succeed in bringing psychological conflicts into the open. One caution for the physician is in order. It is much safer to listen than to talk. Heedlessly giving advice on important emotional factors carries with it many dangers.

BRINGING THE BLOOD PRESSURE DOWN

One of the greatest faults in the management of hypertension in this country has been the emphasis on "bringing the blood pressure down." We must look upon the blood pressure curve in the same way that we do the fever curve in an acute infectious disease and realize that it is just as wrong to fasten our attention on bringing the blood pressure down without understanding and treating the individual who has the blood pressure as to concentrate on reducing the fever. Please understand that I am not objecting to efforts to lower the blood pressure when combined with efforts to understand the patient who has the blood pressure. For the former purpose potassium sulphocyanate is again becoming popular and while I will not take the time to discuss this treatment in detail I must emphasize that it should only be used with strict control for the level of blood cyanate.

We have paid altogether too much attention to physical measurements in hypertensive disease. While it is undeniably true that the size of the heart as determined by the x-ray, the height of the blood pressure as determined by the sphygmomanometer, the condition of the retinal vessels as determined by the ophthalmoscope, and the state of renal function as determined by the various clinicopathologic tests are all absolutely essential to the understanding of the hypertensive individual, they are only the beginning and not the end of the study. We must try to understand the total personality of the individual who has hypertension or who seems destined to develop it. It is the study of emotional factors which may provide us with the key to the successful management of the hypertensive individual.

REFERENCE

(1) Weiss, Edward: Review: Recent Advances in Pathogenesis and Treatment of Hypertension, Psychosomatic Med., 1:180, (January) 1939.

DISCUSSION

DR. VICTOR D. WASHBURN (Wilmington): Mr. President, I think I might say that I was delighted to have the opportunity of bringing Dr. Weiss down today because I am keenly interested in this subject. However, I am not competent to discuss it. I have thoroughly enjoyed the presentation, and can say to him now, for I said just before he began to speak that I didn't know whether I would enjoy his paper, that I have thoroughly enjoyed it and feel that it was well worth my trip here.

Speaking from the standpoint of the urologist, I may say that I have had but limited experience in attempting to discuss this problem with men doing internal medicine. I have not actually attempted to solve the problem of whether or not a given patient, with hypertension, did or did not have renal infection. Since I have not the experience, I am not qualified to discuss it. I do wish to express my pleasure at having heard the paper and to say that from what I have learned I am sure I will be better qualified to go on and study this problem and attempt to be useful to the men who are doing general practice and who are alert to these aspects of hypertension.

DR. E. M. KRIEGER (Wilmington): Mr. Chairman, I enjoyed Dr. Weiss' paper very much. I heard a very competent man say just ten days ago that if we threw the blood pressure apparatus away and spent a half hour with these people, talking with them about their social problems, we would do much better than taking their blood pressure once a week.

I say it was an excellent presentation.

DR. J. D. NILES (Middletown): Mr. President, I would like to ask the importance of

edema if a patient comes into your office and about the only symptom he has that you can see or grasp at your first meeting is that his legs are edematous. That undoubtedly is caused by different things. I would like to know the steps or procedures which Dr. Weiss takes in figuring from that standpoint what the kidneys have to do with it.

DR. WEISS: Dr. Washburn was good enough to say that he enjoyed coming down with me. He didn't know, however whether he was going to enjoy the talk or not. It reminded me of a time when I was out in Youngstown last year and the Jefferson boys had a golf match in the afternoon. The talk was to be in the evening. As we came into the locker house I heard a couple of old fellows behind me say, "Well, I hope he can talk better than he plays golf."

The question of edema has three mechanisms. There is an edema that occurs in acute nephritis, and that is apparently due to a diffuse vascular injury, but there would be no difficulty in the differential diagnosis of that because the signs of acute nephritis are so obvious.

Then there is the edema which occurs in the so-called nephrosis. That has a very different mechanism. It is due to the loss of albumin in the urine. The loss of albumin in the urine depletes the blood proteins, and the depletion of blood proteins alters the quality of the blood for holding fluids within the blood vessels. In other words, the colloidal osmotic pressure of the blood has been reduced. The plasma, therefore, leaks out and we have edema as a result. That is called nephrotic edema.

Then there is the last variety with which you are familiar, cardiac edema, which is due to increased hydrostatic pressure in the blood vessels. The right side of the heart fails. Venous pressure is increased in the pendent portions of the body and fluid leaks out into the tissues.

Obviously, the first variety is easily diagnosed. The last variety is fairly easily diagnosed. One tries to determine whether or not heart failure is present. It is the second one that causes some trouble, and the second one is the much more difficult one to treat, that is, nephrotic edema. The mechanism, of course, is to try to replace those proteins which have been lost through the kidney, and so again we reverse ourselves completely in the treatment of this kidney disease.

Formerly, when a patient with kidney disease had albumin in the urine, one of the first things which was done was to cut out his meat and eggs. We said, "You can't have egg white," for example, "because of albumin in your urine." But the situation is really quite the opposite. He is losing proteins and he needs proteins. You have to give him more instead of less. If you can do that, if you can successfully increase his blood pressure you can increase his colloidal osmotic pressure and his edema will diminish.

Those are the three main forms of edema and that is about the way one would approach them from a diagnostic standpoint.

DR. NILES: Mr. President, I would like to give an experience that I had with edema. I was on a trip to Havana, Cuba, and as we passed Nassau I noticed that my legs and ankles were edematous and I was somewhat startled and surprised, but upon talking with a number of people on that boat that morning, I found that all of them had the same complaint. Some of them were drunk and some of them weren't.

However, we were given a lecture and the ship's doctor brought out this fact: He said, "I suppose a great many of you are startled and surprised that you couldn't get your shoes on this morning. That is due to the climatic change, the sudden change, and perhaps excessive eating." But that didn't answer my question, and I wonder if Dr. Weiss could give the reason for that. That is a fact, what I am stating. At least ninety per cent of the people on that boat complained of edema as we neared Havana.

DR. WEISS: Well, I thought you meant, in the first place, the forms of edema that were associated with vascular disease.

Of course, there are other varieties of edema. There is an edema, for example, which occurs because of diminution in the amount of protein that one eats or a diminution in the ability of the body to absorb that protein. Then there are certain edemas that have to do with increased intravascular tension, which are associated with altitude and

with temperature. I think all of us are familiar with the fact that in the summertime the hands and sometimes the feet are apt to feel a little puffy, and they actually do increase so far as the size of the ankle is concerned, and sometimes there is pitting. That is related at least to cardiac edema, as far as the mechanism is concerned.

I wouldn't know exactly what this edema was that you described. Personally, I am not one who believes that alcohol has any serious effects on the vascular system. I like to believe that it hasn't, and if there was any temporary change that occurred, I think you can put that down to something other than overindulgence in alcohol. Perhaps the ship's doctor was right, that a sudden change in temperature might have done it. I wouldn't know.

DR. H. S. RIGGIN (Seaford): I would like to ask the Doctor one question from the standpoint of the general practitioner: Do you disregard the value of getting rid of the fluid? In other words, if we have edema, and the kidneys are not working, why isn't a diuretic of value?

Of course, I am reluctant to mention these things because I am not qualified to discuss the subject with the Doctor, but from the standpoint of general practice, when we have these cases, we resort to diuretics and we get results momentarily. In other words, we relieve the patients of some symptoms which they have at that time by using diuretics. If we can figure your hypertension together with the kidneys, we know that the kidney is practically the last avenue of escape of the fluids of the body. When these fluids do not escape, and we have edema, we have subjective and objective symptoms which we, as general practitioners, have to handle at the moment. In the old days we used to use—and I presume we do now—hot packs and things of that kind for the purpose of getting rid of some of the fluid. A diuretic has certainly, in my opinion, over a long period of experience, helped us momentarily when we have these cases come up in general practice.

I am not qualified to discuss it from the scientific standpoint, but many of us who are here, who handle these cases as they come up to us in the country or in the city, cannot talk to the patient about scientific things, the reason why we don't use diuretics, and the reason why we don't do this. However, we have used diuretics for years and we have gotten results.

This isn't any criticism in any way. It is simply that I am asking for information because I have done this for years, and if it is not the right thing to do, as time goes on we want to know the new things. However, what are we going to use in place of the diuretics and things to get rid of this fluid which causes these symptoms from which your patient is suffering at the time you see him?

I think this is an opportune time to ask this question, and I would be very grateful if the Doctor would answer it.

DR. I. J. MACCOLLUM (Wyoming): I believe that these foci of infection might be the cause of kidney infection, which might lead to the ischemic condition of the kidney, and that it is an important thing that these foci of infection and overindulgence in alcohol, and in other things, might have a bearing on causing a kidney condition which, after all, is responsible for the hypertension.

DR. WEISS: Well, now I use diuretics in the treatment of edema, too. The point I was trying to make was that one must make a differential diagnosis of edema before he uses a diuretic. For example, if the patient has acute nephritis, you certainly don't want to give him a diuretic, not a mercurial, because you will add to his acute nephritic problem. If he has cardiac edema, on the other hand, a diuretic is just about the best thing you can use, a mercurial diuretic, mercurin, which is a combination of mercury and theophorin, or salyrgan, intravenously, or intramuscularly, will probably do more for the patient than almost anything else you can do. It seems to mobilize the fluid. Sometimes those patients will get rid of quarts of fluid and present a changed picture the following day.

No, indeed, I am very enthusiastic about the use of diuretics. It is only a question of selecting the kind of case for which a diuretic is to be used, and an acute kidney condition, or even a subacute kidney condition is not one of the best in which to use a diuretic. You may do more harm than good.

On the question of focal infection, I don't

believe that focal infection has anything to do with hypertension as such, and I think the increasing evidence, both experimental and clinical, is in that direction. Whether some few forms of actual kidney infection, such as pyelonephritis, or perinephritic abscess, are due to focal infection, I am not prepared to say with certainty. I don't believe so. I think when the final chapter on focal infection is written we will find that we have been overcredulous in America with regard to this concept. It has some value, of that there is no question, but we have sacrificed teeth and tonsils until we could build a monument with them to our American credulity in this respect. I don't believe there is any relationship between vascular disease, kidney disease, and foci of infection. That doesn't mean that if a tooth is diseased or a tonsil is diseased it shouldn't come out; it should, but not because of high blood pressure as its only reason.

In that connection, I might add further that if a patient has advanced kidney disease, with renal impairment, you may do him real harm by operating on him. I have seen patients precipitated into uremia by the wholesale removal of teeth, or yanking out the tonsils. So you have to go easy in recommending operations on patients who have advanced kidney disease or heart disease.

Lastly, on this question of alcohol, I don't believe good alcohol hurts anybody when it is taken in moderation. I had a patient admitted to the hospital yesterday who was spitting blood. When I got his history I was told that he and a gang of his associates had just drunk a quart and a pint of denatured alcohol. It seems that in Philadelphia there is a custom of buying, in certain neighborhoods, a pint of denatured alcohol for sixteen cents, dividing it three ways with water, and then having a crowd polish it off. This fellow spat blood as a result. I asked him if he was sure he wasn't spitting flames. Perhaps it is a new flame-throwing mechanism, but it certainly hit him hard, and I think he is just beginning to recover from it.

I am not speaking of that kind of alcohol. However, I don't believe that ordinary alcohol will do any harm to the hypertensive individual. If you have a hypertensive individual on a dietetic regime, you try to reduce his weight, for example, and yet if you permit him to take a cocktail before dinner, he probably won't pay much attention to his diet, but that is about the only relationship that I can see.

PRESIDENT BARNES: Dr. Weiss, your batting average is still a thousand with us who have heard you before. I wish to thank you in behalf of the Medical Society of Delaware for your very fine presentation.

GENITO-URINARY DISEASE AND DIAGNOSIS*

ERVIN L. STAMBAUGH, M. D.,
Lewes, Del.

It is desirable to say at the outset that we do not intend to cover the entire text of urological diagnosis in one paper. From an anatomical and pathological standpoint, we would have to include diseases of the kidneys, ureters, bladder, and urethra in both male and female, as well as prostate, seminal vesicles and external genitalia in the male. Also, we would have to discuss mechanical conditions; for example, calculi and injuries, acute and chronic inflammations, and benign and malignant neoplasms of the entire urinary tract. Believing there is a real need for accurate diagnosis, we wish to emphasize the importance of a urological examination in any case of obvious or obscure symptoms referable to the genito-urinary tract.

A great many cases in urology require two or three days of investigation before we are able to make a final diagnosis. Before the advent of x-ray and cystoscopy physicians were limited in their study of patients. A pain in the back was unqualified "kidney trouble." To their patients, this was as final a diagnosis as to say they had pneumonia. If the diagnosis of kidney trouble did not work out clinically, they had recourse to lumbago as another working diagnosis.

In presenting a schema of investigation, we wish to emphasize two points: (1) the part that a good office diagnosis plays in the final diagnosis; and (2) the help that can be expected with the more modern x-ray and cystoscopic examinations.

*Read before the Sussex County Medical Society, Georgetown, April 10, 1941.

HISTORY

Office diagnosis considers first of all a history of symptoms. These include pain, referred to any part of the genito-urinary tract, strangury, frequency of urination or obstruction, and, finally, alterations in the appearance of the urine noted by the patient himself; for example, hematuria.

Symptomless hematuria is a complaint calling for urgent diagnosis. Nine out of ten times it precludes accurate diagnosis in the office, and such patients should be forthwith asked to submit to x-ray and cystoscopic examination. It is almost as hazardous to put a patient, who is complaining of hematuria, to bed with an ice bag, as it is to administer morphia to a patient with abdominal pain as long as any doubt remains as to diagnosis. It is characteristic of bleeding of neoplasm of the bladder or upper urinary tract to be intermittent, and once the bleeding has stopped, it may not occur again for a long time.

PHYSICAL EXAMINATION

The next step should be a complete physical examination. It must include inspection and palpation of the lower urinary tract for tumors or enlargement of the scrotum, vesical distention and renal or abdominal masses. Venereal conditions, so-called, should be established or ruled out. A patient, recently referred from a venereal clinic as non-venereal, presented the following: (1) tuberculosis of the epididymis and vas; (2) seminal vesicle extension on rectal examination; (3) cystoscopic appearance of involvement of the bladder by gaping of the right ureteral orifice. tuberculous ulceration and tubercle formation; (4) unilateral, possibly bilateral renal tuberculosis on intravenous urography; and, finally (5) x-ray evidence of miliary tuberculosis of the lungs. And, yet, examination of the epididymis was sufficient evidence of genito-urinary tuberculosis. Fully 90% of such cases as the above will show a variable degree of enlargement of the epididymis and vas.

A suprapubic tumor with the characteristics of a distended bladder is the most important sign of lower urinary tract obstruction. Ruling out urethral stricture in the male by passing sounds, we are left with the diagnosis of vesical neck obstruction.

Major causes of vesical neck obstruction in the male are: (1) cancer of the prostate, 5-10%; (2) median bar, 15%; and (3) benign hypertrophy of the prostate, 60% plus. Minor causes are: (1) tuberculosis of the prostate; (2) vesical and prostatic stones; (3) benign and malignant tumors of the bladder; (4) diverticulum; and (5) foreign bodies. The above minor causes, with exceptions, apply to vesical neck obstruction in the female and in children.

No physical examination in urology is complete without a rectal examination. We heard a surgeon, in discussing a paper of Buie's, ask the truth of the report that every patient that entered the Mayo Clinic was subjected to proctoscopic examination. In denying the report, Buie stressed the fact that nearly every patient did get a rectal examination. All will agree that no examination in their experience has been as fruitful of "lucky" information as a rectal examination.

Finally, a good office diagnosis should develop physical signs and history of concurrent disease such as uremia, pulmonary disease, cardio-vascular disease, or syphilis. Obtaining this information is perhaps not as important in the diagnosis as it is in the subsequent management of individual cases.

URINE

In the schema of diagnosis we urge the microscopic examination of the urine as part of every routine examination. But there is even a refinement on the physician's request of "send your urine in." Referring again to hematuria, to distinguish infection from stone or neoplasm, it merely means culturing an aseptically drawn urine. A slide of the stained sediment of a sterile urine may be done as an alternative. Also, in doubtful cases of genito-urinary tuberculosis guinea pig inoculation of a urinary sediment is of the greatest value. With the advent of the sulfonamide group of drugs in the treatment of diseases of all systems, the examination of urine must include a search for sulfonamide crystals. The crystals may be so abundant as to "stampede" in the pelvis and ureter, causing renal colic or finally coalesce into calculi.

X-RAY

For awhile, we want to consider those cases in which help can be expected with x-ray methods. Good x-ray technique will not compensate for haphazard preparation. Our routine is to give three ounces of castor oil the evening before, followed with an enema in the morning. No breakfast is given the morning of the examination.

Several years ago, Lower[1] wrote of a group of cases as silent lesions of the upper urinary tract. He referred to them as silent because they did not produce any outstanding subjective symptoms which obviously pertained to the kidneys or ureters. Such symptoms as nausea, belching, vague abdominal pains and constipation are frequently associated with kidney conditions. Backache may direct our attention to the neuro-muscular system. It is in these cases that a searching examination with the cystoscope, with the aid of x-rays, would be made to rule out or establish a lesion in the urinary tract.

The following case histories are offered to illustrate diagnostic difficulties:

Case I. A female, aged 50, was admitted to the hospital with complaints of constipation, pain over lower abdomen, gaseous distention, and mucous in stools. Patient had "neuritis" pains in shoulder and left lumbar region.

In her past history there was a record of intestinal trouble, no bowel movement without laxatives or enemas, and intestinal gas over a period of five years. She had had G. I. x-rays, electrocardiogram, and a B. M. R. made elsewhere. These were negative. No x-rays of the genito-urinary tract were taken.

In the physical examination, it was brought out that she had slight tenderness in the left costovertebral angle.

Urine examinations: Several were made. Albumen was positive. Pus cells and red blood cells were found in variable numbers, from 15-20 white blood cells, and from 3-5 to as high as 75 red blood cells per high power field.

X-ray showed a pelvic type of urinary calculus on the left side. An excretory pyelogram revealed secondary hydronephrosis and hydroureter of moderate degree.

Comment: We had a great deal of difficulty in getting this patient away from her G. I. diagnosis—vague as it was—of colitis.

In this case, the x-ray told the story, but so did the persistence of red blood cells in the urine.

At operation, the kidney substance was reduced to a sac by a large hydronephrosis. Nephrectomy had to be done. A few years ago, this kidney could probably have been saved. We have no doubt that she will have enough kidney substance left, but no one will deny that a second kidney is a good thing to have.

Case II. Briefly, this patient was a man of 57 years, whose chief complaint was epigastric distress. In his past history he had had several attacks of renal colic, on occasion passing several calculi. For the past four years, he presented no symptoms referable to the kidney. For the epigastric distress, his physician was prescribing dilute hydrochloric acid. X-rays had not been taken.

Comment: The true condition of multiple renal calculi was diagnosed by pyelographic and cystoscopic methods.

CYSTOSCOPY

In the absence of contraindications, most urological patients should be subjected to cystoscopy. In many it will prove to be a most valuable aid to diagnosis; in a few it will be superfluous. Contraindications to cystoscopy are not within the scope of this paper, but in brief they are the acute infections of the lower urinary tract, in which it seems better to postpone the procedure until a less acute stage. Also, when the diagnosis is clear, this examination should be omitted in prostatism with uremic symptoms.

At the first cystoscopy, or a subsequent one, the ureters are catheterized. Separate urines are routinely examined microscopically and culturally. Dye, such as indigocarmine, function is determined.

If we define urography as the x-ray demonstration of any part of the urinary tract after opaque media is injected through ureteral catheter or intravenous routes, we have then an important means of (1) demonstrating the pelvic outline; (2) detecting congenital abnormalities; (3) determining the course or obstruction of the ureter; and (4) learning the relationship of extraurinary pathology. Since

1929, intravenous urography has provided an alternate method, particularly applicable to cases where cystoscopy and ureteral catherterization are difficult, impossible, or undesirable. It finds an important application, particularly in children.

Conclusions: In conclusion, we want to emphasize the importance of a good history, a complete physical examination, including rectal examination, routine urine examinations, and a slide of the stained sediment. X-ray and cystoscopic investigations are recommended in any case of obscure symptoms referable in to the genito-urinary tract.

Beebe Hospital.

REFERENCE

1. Lower, W. E.: Silent Lesions of the Upper Urinary Tract, Proc. Interst. Grad. M. A., 1930.

RE: BUNDLES FOR BRITAIN

As much as we individually might be in sympathy with the "Bundles for Britain" movement, one recent phase of it hardly has our approval.

At several points in the country there has been a movement to collect the samples left by pharmaceutical detail men in physicians' offices and include them in the shipments for British relief. This is an expensive and uncontrolled way of supplying pharmaceutical products.

Most all of the pharmaceutical manufacturers have individually donated supplies with vitamin capsules and other needed pharmaceutical products to the British relief at no charge.

The packaging of a sample increases the cost and if these samples are collected and sent to Britain, then the purpose for which they were intended, that is, for the use of physicians, is not accomplished, and the heterogenous material that reaches British relief probably would have little value. Many samples left physicians would be dangerous if used indiscriminately without the advice of a physician.

In some cases individual City and County Medical Societies have been asked to cooperate with the collection of these samples. It is our opinion that such cooperation should be refused for the obvious reasons stated.

MISCELLANEOUS

American Medical Association Cleveland: June 2-6, 1941

The Ninety-second Annual Session of the A. M. A. will be held in Cleveland the first week in June. The House of Delegates will convene at 10 a. m., Monday, June 2. Delaware will be represented by her only delegate, Dr. Laurence L. Pritchett, of Milford, whose reports of previous meetings have been detailed and instructive. The House will be directed by its able speaker, Dr. H. H. Shoulders, of Nashville. Dr. Nathan B. Van Etten, of New York, is the retiring President, and Dr. Frank H. Lahey, of Boston, is the incoming President.

The scientific program, as arranged for the various sections, is a broad and interesting one. The only member from Delaware who is on the program is Dr. M. A. Tarumianz, of Farnhurst, who will participate in a panel discussion on neurosurgical treatment of certain abnormal states, on Thursday afternoon, June 5, before the Section on Nervous and Mental Diseases. This discussion will center on subcortical lobotomy for dementia praecox, agitated depression, etc. Dr. Tarumianz will relate the experience of the Delaware State Hospital, where numerous lobotomies have been performed, with encouraging results.

EDITORIAL

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POISED FOR THE KILL

As yet it is too early to analyze, evaluate, forecast or even hazard a guess as to just what the decision of the Federal court against the American Medical Association and the Medical Society of the District of Columbia will mean to the medical profession throughout the nation. For one thing, definite statements should await the results of the appeal by the American Medical Association. However, one thing is certain: the physicians who made up the original committee of 430—you know the names of some of their leaders, Cabot, Peters, Sigerist, Warbasse, Winslow, Michael Davis—are poised for the kill. Over the radio in the air, from speakers' platforms on the ground, and from their headquarters in New York they are on the alert for "the new order" in American Medicine just as certain leaders of the minority groups in Europe today are awaiting for what they term "the new order" of the world.

Here's what happened and is happening now to make us wary of this group who want to change traditional medical practices. While the American Medical Association trial was still in progress, the Cabot-Peters axis partners through an organization known as the "Bureau of Cooperative Medicine" began to step up their propaganda activities, to tell the whole wide world of the beauties of cooperative medicine. They supply the press with a never-ending stream of bulletins; a monthly called "*Medical Care* — Economic and Social Aspects of Health Service" puts out its first issue and Michael Davis, of "The Committee on the Cost of Medical Care" fame, markets a new book entitled "America Organizes Medicine." The news bulletins from the Bureau carry well-prepared briefs on the A. M. A. trial. Then one day comes a communique stating that the name of the Bureau of Cooperative Medicine is changed to "Medical Administration Service." The purposes of "Medical Administrative Service" will be to act as "both a national clearing house for the collection and dissemination of experiences and a consulting service which will advise on the basis of its aggregate experience. Experts in various fields are an integral part of this new organization."

Immediately we wonder what information such an organization could have that is not already available through the Bureau of Medical Economics of the American Medical Association whose experts are in intimate touch with all situations, experiments in health insurance and methods being planned, or actually in use in the various communities throughout the entire country.

The natural question arises, why the change in name? Can it mean that John Peters, head feuhrer and duce, and Hugh Cabot as minister of propaganda and public

(*Continued on Page xviii*)

BOOK REVIEWS

The Merck Manual of Therapeutics and Materia Medica 7th Edition. Pp. 1436. Cloth. Price, $2.00. Rahway: N. J.: Merck & Company, 1940.

How this Manual has grown! We remember an earlier edition of about 500 pages, but this new one is far beyond the former *vade mecum* stage of its ancestors. It contains five parts: (1) Therapeutic indications, which is a text book of medicine in brief form, with numerous prescriptions; (2) poisoning and its treatment; (3) dose table; (4) materia medica; (5) miscellany. Part One, to which twelve new chapters have been added, is exceptionally up to date, and embraces 256 clinical entities. The index is quite ample. The size of the page and of the type has been slightly enlarged, and the paper stock is a bit heavier, yet the book still remains a handy volume, though no longer of the pocket variety. In fact, it contains more information than any other book its size that we know of.

The new Merck Manual is one of the most reliable of its kind available today; it certainly is a whale of a buy for two bucks.

"The Essentials of Applied Medical Laboratory Technic." By J. M. Feder, M. D., Director of Laboratory, Anderson County Hospital, Anderson, S. C. Pp. 241. Cloth. Price —. Charlotte, N. C.: Charlotte Medical Press, 1940.

The contents of this unique book is best described by the subtitle, "Details of How to Build and Conduct an Office or Small Hospital Laboratory at Small Cost." The technical methods are sound and the suggested improvisations of apparatus may be made by any person who can use simple carpenter's tools. This book may well solve the problems of laboratory equipment for small hospitals with limited income, and for physicians who do not wish to mortgage five year's income in equipping an office and laboratory.

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1. Knight, F., and Shelanski, H. A., "Treatment of Acute Anterior Urethritis with Silver Picrate," Am. J. Syph., Gon. & Ven. Dis., 23, 201 (March), 1939.

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POISED FOR THE KILL

(*Continued from Page 83*)

enlightenment, believe as do some other great dictators who seek to destroy our democratic world that the time is ripe to supplement the activities of the American Medical Association, or perhaps like other great visionaries that now is the time to get set for the kill and take over the American Medical Association?

Editorial, *J. Ind., S. M. A.*, May, 1941.

THAT VERDICT

Among the interesting comments on the recent paradoxical verdict *against* the organization but *for* the individuals composing that organization (!) is this from the Chicago Tribune:

> The anti-trust conviction may impress upon the members of the A. M. A. that when they organized they took out the wrong kind of a charter. They should have applied to William Green or John L. Lewis. So equipped, they would not have been reduced to refusing to practice in the same hospitals with a physician who signed up with Group Health. Dr. Morris Fishbein could just have gone around some evening and broken the wrong guy's fingers with a blackjack, an operation that does a surgeon no more good than it does a musician, and Mr. Justice Frankfurter would have told Thurman Arnold not to get himself all wrought up over a passing moment of animal exuberance.
>
> A good broad A. F. L. or C. I. O. charter would solve a lot of the medical profession's economic problems. Its members would not have to worry about overproduction of doctors. They could just close their membership rolls and have some of their members, sitting on the state and local examining boards, prosecute the newcomers for practicing without a license.
>
> Draft boards wouldn't be asking physicians to give their services free for examination of the draftees. All the chest thumping in charity wards would be done at the union scale and any non-union medico who tried to cut in on the business would have to pay a $1,000 initiation fee. Ladies expecting offspring would have to be careful that the labor pains did not start after 4 p. m. on a Friday; otherwise papa would have to pay double time for a week-end delivery.
>
> The medical union might be able to take on a number of profitable activities that A. M. A. members now deny themselves, such as performing abortions, or, for a suitable fee, slipping a dose from the black bottle to millionaires whose heirs were growing impatient. While such activities might arouse public protest, the union docs should be sure that President Green would not bother them. That would be interfering with their autonomy.
>
> The dear old Tribune may have something there at that. Dear John, when can you send the muscle men?

Editorial, *Southwest Med.*, April, 1941.

We are beginning to wonder what is wrong with "official" Washington. We had just about become tuned in to the anti-trust law charge against the American Medical Association, together with numerous other indictments aimed against other organizations and corporations when the news came that Eli Lilly and Company, Sharp and Dohme, and E. R. Squibb and Sons had been indicted because of their alleged control of the manufacture and control of insulin. The early history of insulin is known to most medical men; the fact that the process of manufacture was patented is known to all, as well as the fact that the preparation and the marketing of the product was limited to a very few pharmaceutical companies. To all this the medical profession was in accord, since it assured us of a safe product and at a fair price. It is, of course, true that in the early days insulin was an expensive luxury, but since then, according to Eli Lilly, there have been thirteen reductions in price. Today, insulin costs the consumer just three and one-half per cent what it did when it first was introduced. We need not launch into a discussion of the merits of the matter, save to say that had there been no exercise of control over this product the market would have been flooded with an inferior drug. One can but wonder what might have happened had certain of the vitamins been thrown into an open market. As it is now, vitamins are well under control and the prescriber is assured that when he orders certain of the products for his patient, that is just what the patient is going to get. No stretch of the imagination is required to think of the lives that might have been saved had sulfanilamide and sulfathiazole been patented, thus placing them under some degree of manufacturing control. Again, one wonders about the patent medicines; they seem to be under control and the manufacturers thereof exempt from federal prosecutions.

Editorial, *J. Ind. S. M. A.*, May, 1941.